```
=> s (density enhanced polypeptide-1 or DEP_1)
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FULL ESTIMATED COST                                 0.42       0.42

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=> s (density enhanced polypeptide-1 or DEP-1)
L1          318 (DENSITY ENHANCED POLYPEPTIDE-1 OR DEP-1)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2          130 DUP REM L1 (188 DUPLICATES REMOVED)

=> s l2 and signal transduction
L3           19 L2 AND SIGNAL TRANSDUCTION

=> s l3 and DEP-1 substrate polypeptide
L4            0 L3 AND DEP-1 SUBSTRATE POLYPEPTIDE

=> d l3 1-19 ibib ab

L3   ANSWER 1 OF 19     MEDLINE on STN
ACCESSION NUMBER:   2005515175     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 16114069
TITLE:              Genes critical to vasculogenesis as defined by systematic
                    analysis of vascular defects in knockout mice.
AUTHOR:             Argraves W Scott; Drake Christopher J
CORPORATE SOURCE:   Department of Cell Biology and Anatomy, Medical University
                    of South Carolina, Charleston, South Carolina 29425, USA..
                    argraves@musc.edu
CONTRACT NUMBER:    HL52813 (NHLBI)
                    HL57375 (NHLBI)
                    HL61873 (NHLBI)
SOURCE:             The anatomical record. Part A, Discoveries in molecular,
                    cellular, and evolutionary biology, (2005 Oct) Vol. 286,
                    No. 2, pp. 875-84.
                    Journal code: 101234285. ISSN: 1552-4884.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
```

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 29 Sep 2005
Last Updated on STN: 22 Dec 2005
Entered Medline: 20 Dec 2005

AB To identify genes important to the process of vasculogenesis, we evaluated embryonic vascular anomalies from 100 mouse knockout studies using a novel meta-analysis approach. By applying this method, termed approach for ranking of embryonic vascular anomalies (AREVA), rank scores were calculated for each knockout based on the occurrence of vascular defects during periods of vasculogenesis in specific embryonic regions. As a result, 12 genes (fibronectin, VEGFR-1/Flt-1, VEGFR-2/Flk-1, alpha 5 integrin, Tek/Tie2, VE-cadherin, VEGFA, connexin 45, ShcA, cytochrome P450 reductase, CD148/DEP-1, and EphrinB2) were determined to play critical roles in vasculogenesis. Functional categorization of these genes revealed the fundamental importance of VEGF signaling since 10 of the 12 genes (fibronectin, VEGFR-1/Flt-1, VEGFR-2/Flk-1, alpha 5 integrin, VE-cadherin, VEGFA, ShcA, cytochrome P450 reductase, CD148/ DEP-1, and EphrinB2) relate to this pathway. Furthermore, the findings highlight a potential network for regulating VEGF signaling involving integration of fibronectin, EphrinB2, Tie2, and connexin 45 signaling pathways via the ShcA/Ras/Raf/Mek/Erk cascade. In addition to retrospective application of AREVA as done herein, AREVA can be used prospectively to determine the relevancy to vasculogenesis of newly inactivated genes.

L3 ANSWER 2 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2005290599 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15901674
TITLE: The C. elegans homolog of the mammalian tumor suppressor Dep-1/Scc1 inhibits EGFR signaling to regulate binary cell fate decisions.
AUTHOR: Berset Thomas A; Hoier Erika Frohli; Hajnal Alex
CORPORATE SOURCE: Institute of Zoology, University of Zurich, Switzerland.
SOURCE: Genes & development, (2005 Jun 1) Vol. 19, No. 11, pp. 1328-40. Electronic Publication: 2005-05-18.
Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 7 Jun 2005
Last Updated on STN: 27 Sep 2005
Entered Medline: 26 Sep 2005

AB Protein phosphorylation by kinases and the subsequent dephosphorylation by phosphatases are key mechanisms that regulate intracellular signal transduction during development. Here, we report the identification of the receptor protein tyrosine phosphatase DEP-1 as a negative regulator of the Caenorhabditis elegans EGF receptor. DEP-1 amplifies in the developing vulva and the excretory system the small differences in the amount of EGF signal received by equivalent precursor cells to achieve binary cell fate decisions. During vulval development, DEP-1 inhibits EGFR signaling in the secondary cell lineage in parallel with the NOTCH-mediated lateral inhibition, while EGFR signaling simultaneously down-regulates DEP-1 and NOTCH expression in the primary cell lineage. This regulatory network of inhibitors results in the full activation of the EGFR/RAS/MAPK pathway in the primary vulval cells and at the same time keeps the EGFR/RAS/MAPK pathway inactive in the adjacent secondary cells. Mammalian Dep-1/Scc1 functions as a tumor-suppressor gene in the intestinal epithelium. Thus, mutations in human Dep-1 may promote tumor formation through a hyperactivation of the EGF receptor.

L3 ANSWER 3 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2004029765 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14709717
TITLE: The tyrosine phosphatase DEP-1 induces cytoskeletal rearrangements, aberrant cell-substratum interactions and a reduction in cell proliferation.
AUTHOR: Kellie Stuart; Craggs Graham; Bird Ian N; Jones Gareth E
CORPORATE SOURCE: School of Molecular and Microbial Sciences, Institute for Molecular Bioscience and CRC for Chronic Inflammatory Diseases, University of Queensland, Brisbane, QLD 4072, Australia.. s.kellie@mailbox.uq.edu.au
SOURCE: Journal of cell science, (2004 Feb 1) Vol. 117, No. Pt 4, pp. 609-18. Electronic Publication: 2004-01-06. Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200409
ENTRY DATE: Entered STN: 21 Jan 2004
Last Updated on STN: 30 Sep 2004
Entered Medline: 29 Sep 2004

AB The receptor protein tyrosine phosphatase density-enhanced phosphatase-1 (DEP-1) has been implicated in aberrant cancer cell growth and immune cell function, however, its function within cells has yet to be properly elucidated. To investigate the cellular function of DEP-1, stable cell lines inducibly expressing DEP-1 were generated. Induction of DEP-1 expression was found to decrease PDGF-stimulated tyrosine phosphorylation of a number of cellular proteins including the PDGF receptor, and to inhibit growth factor-stimulated phosphorylation of components of the MAPK pathway, indicating that DEP-1 antagonised PDGF receptor signalling. This was supported by data showing that DEP-1 expression resulted in a reduction in cell proliferation. DEP-1-expressing cells had fewer actin-containing microfilament bundles, reduced vinculin and paxillin-containing adhesion plaques, and were defective in interactions with fibronectin. Defective cell-substratum adhesion correlated with lack of activation of FAK in DEP-1-expressing cells. Time-lapse interference reflection microscopy of live cells revealed that although small focal contacts at the leading edge were generated in DEP-1-expressing cells, they failed to mature into stable focal adhesions, as found in control cells. Further motility analysis revealed that DEP-1-expressing cells retained limited random motility, but showed no chemotaxis towards a gradient of PDGF. In addition, cell-cell contacts were disrupted, with a change in the localisation of cadherin from discrete areas of cell-cell contact to large areas of membrane interaction, and there was a parallel redistribution of beta-catenin. These results demonstrate that DEP-1 is a negative regulator of cell proliferation, cell-substratum contacts, motility and chemotaxis in fibroblasts.

L3 ANSWER 4 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2003304944 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12833140
TITLE: The protein-tyrosine phosphatase DEP-1 modulates growth factor-stimulated cell migration and cell-matrix adhesion.
AUTHOR: Jandt Enrico; Denner Karsten; Kovalenko Marina; Ostman Arne; Bohmer Frank-D
CORPORATE SOURCE: Research Unit Molecular Cell Biology, Medical Faculty, Friedrich Schiller University, Drackendorfer str 1, D-07747 Jena, Germany.
SOURCE: Oncogene, (2003 Jul 3) Vol. 22, No. 27, pp. 4175-85. Journal code: 8711562. ISSN: 0950-9232.

PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 1 Jul 2003
Last Updated on STN: 2 Aug 2003
Entered Medline: 1 Aug 2003

AB Density-enhanced protein-tyrosine phosphatase-1 (DEP-1 also CD148) is a transmembrane molecule with a single intracellular PTP domain. It has recently been proposed to function as a tumor suppressor. We have previously shown that DEP-1 dephosphorylates the activated platelet-derived growth factor (PDGF) beta-receptor in a site-selective manner (Kovalenko et al. (2000). J. Biol. Chem. 275, 16219-16226). We analysed cell lines with inducible DEP-1 expression for cellular functions of DEP-1. Several aspects of PDGFbeta-receptor signaling were negatively affected by DEP-1 expression. These include PDGF-stimulated activation of inositol trisphosphate formation, Erk1/2, p21Ras, and Src. Activation of receptor-associated phosphoinositide-3 kinase activity and of Akt/PKB were weakly attenuated at early time points of stimulation. Inhibition of PDGF-stimulated signaling depended on DEP-1 catalytic activity. Importantly, DEP-1 inhibited PDGF-stimulated cell migration. The catalytically inactive DEP-1 C1239S variant enhanced cell migration and PDGF-stimulated Erk1/2 activation, suggesting a dominant negative interference with endogenous DEP-1. In contrast to cell migration, cell-substrate adhesion was promoted by active DEP-1 and delayed or suppressed by DEP-1 C1239S, correlating with positive effects of DEP-1 on adhesion-stimulated Src kinase. We propose that negative regulation of growth-factor stimulated cell migration and promotion of cell-matrix adhesion may be related to the function of DEP-1 as tumor suppressor.

L3 ANSWER 5 OF 19 MEDLINE on STN
ACCESSION NUMBER: 2000066871 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10599888
TITLE: Expression of the membrane protein tyrosine phosphatase CD148 in human tissues.
AUTHOR: Autschbach F; Palou E; Mechtersheimer G; Rohr C; Pirotto F; Gassler N; Otto H F; Schraven B; Gaya A
CORPORATE SOURCE: Institute of Pathology, Heidelberg University, Germany.
SOURCE: Tissue antigens, (1999 Nov) Vol. 54, No. 5, pp. 485-98. Journal code: 0331072. ISSN: 0001-2815.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 24 Jan 2000
Last Updated on STN: 24 Jan 2000
Entered Medline: 11 Jan 2000

AB CD148, a receptor-like protein tyrosine phosphatase also known as HPTP-eta/DEP-1, is involved in signal transduction in leucocytes and is thought to contribute to mechanisms of cellular differentiation. We have investigated the in situ expression of CD148 in various fresh-frozen tissues by immunohistology and analyzed its expression on subpopulations of activated peripheral blood leucocytes by flow cytometry. In lymphoid organs, CD148 was found to be widely expressed on B and T cells, granulocytes, macrophages, certain dendritic cells as well as mature thymocytes. The cellular level of CD148 was increased after in vitro activation of peripheral blood leucocytes. Comparative analysis of tissue samples from normal gut and from patients with active Crohn's disease showed that leucocytes expressing CD148 are

significantly upregulated in inflamed tissues and that a subset of these cells co-express the activation marker CD25. In non-lymphoid tissues, CD148 was found to be present on many epithelial cell types with glandular and/or endocrine differentiation as well as on fibrocytes, melanocytes and Schwann cells. CD148 expression was maintained also in malignant counterparts of such tissues. However, a marked loss of CD148 immunoreactivity was apparent in some of the investigated high-grade carcinomas. In summary, our results confirm a role of CD148 as a leucocyte activation marker. Among non-hematopoietic cells, CD148 is expressed by characteristic types of epithelial and non-epithelial cells. Downregulation of CD148 might promote dedifferentiation and autonomous growth of such cells in malignant tumors.

L3 ANSWER 6 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1999403084 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10473595
TITLE: Inactivation of protein-tyrosine phosphatases as mechanism of UV-induced signal transduction.
AUTHOR: Gross S; Knebel A; Tenev T; Neininger A; Gaestel M; Herrlich P; Bohmer F D
CORPORATE SOURCE: Research Unit "Molecular Cell Biology," Klinikum der Friedrich Schiller Universitat Jena, Drackendorfer Strasse 1, D-07747 Jena, Germany.
SOURCE: The Journal of biological chemistry, (1999 Sep 10) Vol. 274, No. 37, pp. 26378-86.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 14 Oct 1999
Last Updated on STN: 14 Oct 1999
Entered Medline: 7 Oct 1999
AB UV irradiation of cells causes ligand-independent activation of receptor tyrosine kinases. On the basis of dephosphorylation kinetics, UV-induced inactivation of receptor-directed tyrosine phosphatases (PTP) has been proposed as the mechanism of receptor activation (Knebel, A., Rahmsdorf, H. J., Ullrich, A., and Herrlich, P. (1996) EMBO J. 15, 5314-5325). Here we show that four defined protein-tyrosine phosphatases (PTPs), SHP-1, RPTPalpha, RPTPsigma, and DEP-1, are partially inactivated upon UV irradiation of PTP-overexpressing cells. The dephosphorylation of coexpressed platelet-derived growth factor beta (PDGFbeta) receptor by RPTPalpha is inhibited upon UV irradiation. UV converts RPTPalpha into a substrate-trapping enzyme which can coprecipitate PDGFbeta receptor, similarly to the PTP mutant at the active-center cysteine: C433S. In agreement with the proposed mechanism that inactivation of PTPs accounts for receptor tyrosine kinase activation, no evidence for a UV-induced receptor cross-linking could be obtained in PDGFbeta receptor-enriched membrane micelle preparations and in PDGFbeta receptor overexpressing 293 cells. The intrinsic activity of PDGFbeta receptor kinase was required for the UV-induced enhancement of receptor phosphorylation, but was not changed upon UV irradiation. The data support a mechanism of UV-induced signal transduction involving inactivation of PTPs through an unknown reactive intermediate that oxidizes the conserved cysteine in the active sites of PTPs.

L3 ANSWER 7 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1998200586 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9531590
TITLE: CD148 is a membrane protein tyrosine phosphatase present in all hematopoietic lineages and is involved in signal transduction on lymphocytes.
AUTHOR: de la Fuente-Garcia M A; Nicolas J M; Freed J H; Palou E;

Thomas A P; Vilella R; Vives J; Gaya A
CORPORATE SOURCE: Servei d'Immunologia, Servei de Medicina Interna, Hospital Clinic, Barcelona, Spain.
SOURCE: Blood, (1998 Apr 15) Vol. 91, No. 8, pp. 2800-9.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 20 May 1998
Last Updated on STN: 20 May 1998
Entered Medline: 12 May 1998

AB Evidence is presented showing that a protein tyrosine phosphatase different from CD45 is present on the membrane of human hematopoietic cells. The molecule recognized by the monoclonal antibody 143-41, which has been classified as CD148 in the VI International Workshop on Leukocyte Differentiation Antigens, was immunopurified and sequenced. The sequence obtained from N-terminus as well as from two different CNBr-digested peptides showed a close identity with a previously described tyrosine phosphatase named HPTP-eta/DEP-1. CD148 is present on all hematopoietic lineages, being expressed with higher intensity on granulocytes than on monocytes and lymphocytes. Interestingly, whereas it is clearly present on peripheral blood lymphocytes, it is poorly expressed on different lymphoid cell lines of T and B origin. When this protein tyrosine phosphatase was cocrosslinked with CD3, an inhibition of the normally observed calcium mobilization was observed. This inhibition correlates with a decrease in phospholipase C-gamma (PLC-gamma) phosphorylation and is similar to the one observed with CD45. In addition, it is shown that the crosslinking of the CD148 alone is also able to induce an increase in [Ca2+]i. This increase is abolished in the presence of genistein and by cocrosslinking with CD45. These data, together with the induction of tyrosine phosphorylation on several substrates, including PLC-gamma, after CD148 crosslinking, suggest the involvement of a tyrosine kinase-based signaling pathway in this process. In conclusion, the data presented show that CD148 corresponds to a previously described protein tyrosine phosphatase HPTP-eta/DEP-1 and that this molecule is involved in signal transduction in lymphocytes.

L3 ANSWER 8 OF 19 MEDLINE on STN
ACCESSION NUMBER: 1998124491 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9464844
TITLE: Functional characterization of receptor-type protein tyrosine phosphatase CD148 (HPTP eta/DEP-1) in Fc gamma receptor IIa signal transduction of human neutrophils.
AUTHOR: Hundt M; Schmidt R E
CORPORATE SOURCE: Medizinische Hochschule Hannover, Abteilung Klinische Immunologie, Germany.. Hundt.Matthias@MH-Hannover.de
SOURCE: European journal of immunology, (1997 Dec) Vol. 27, No. 12, pp. 3532-5.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 6 Mar 1998
Last Updated on STN: 6 Mar 1998
Entered Medline: 20 Feb 1998

AB Activation of the 40-kDa low-affinity receptor for IgG (Fc gammaRIIa, CD32) leads to tyrosine phosphorylation, increase of cytosolic free calcium concentration ([Ca2+]i), and production of superoxide anions (O2-) in neutrophils (PMN). It has been established that protein tyrosine

kinases (PTK) and phosphatases (PTP) are essential for the regulation of intracellular signaling. CD45 is a type I receptor-type protein tyrosine phosphatase (RPTP) with two PTP domains. Recently it has been demonstrated that co-cross-linking of CD45 modulates the signal transduction pathway of Fc gammaRIIa in PMN. In contrast, the functional characteristics of CD148 (HPTP eta/DEP-1), a new RPTP with only one PTP domain, is unknown. CD148 is expressed on PMN in slightly lower density than CD45, and in higher density than on lymphocytes. [Ca2+]i measured with fluo-3-loaded PMN by flow cytometry and O2- production determined by lucigenin-dependent chemiluminescence were inhibited by co-cross-linking of CD45 with Fc gammaRIIa in comparison to isotype control monoclonal antibody (mAb). In contrast, pre-incubation with CD148 mAb 143-41 abolished O2- generation, but did not inhibit [Ca2+]i rise. In summary, both clustered human RPTP, CD45 and CD148, inhibit Fc gammaRIIa-induced O2- production in PMN, but they differ in regulation of [Ca2+]i. Therefore, it is suggested that co-cross-linking of Fc gammaRII with CD45 and CD148 leads to dephosphorylation of different substrates. These distinct functional capacities may be important for differential regulation of Fc gammaR signaling by currently unknown ligands.

L3 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:467993 HCAPLUS
DOCUMENT NUMBER: 141:35315
TITLE: DEP-1 receptor protein tyrosine phosphatase interacting proteins and related methods
INVENTOR(S): Palka-Hamblin, Helena L.; Tonks, Nicholas K.
PATENT ASSIGNEE(S): Cold Spring Harbor Laboratory, USA
SOURCE: PCT Int. Appl., 130 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004048549	A2	20040610	WO 2003-US38089	20031126
WO 2004048549	A3	20060216		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2503736	AA	20040610	CA 2003-2503736	20031126
AU 2003298761	A1	20040618	AU 2003-298761	20031126
US 2004161821	A1	20040819	US 2003-723606	20031126
EP 1576139	A2	20050921	EP 2003-796519	20031126
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: US 2002-429746P P 20021126
WO 2003-US38089 W 20031126

AB Proteins are identified from human breast tumor cell lines (MDA-MB-231, T-47D and T-47D/Met) that interact specifically with the substrate-trapping mutant form of D. Enhanced Phosphatase-1 (DEP -1). These proteins include the functional component p120 catenin (p120ctn), the adaptor protein Gab 1, and the HGF/SF receptor Met. The invention relates to isolated complexes comprising DEP-1 polypeptides in specific assocn. with Met, Gab 1, or p120ctn, identified herein as DEP-1 substrate polypeptides.

Screening assays for agents that alter DEP-1 interaction with DEP-1 substrate polypeptides are also disclosed, as are methods for altering biol. signals in cells that are transduced via DEP-1 pathways.

L3 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:717658 HCAPLUS
DOCUMENT NUMBER: 139:242287
TITLE: Engineering protein tyrosine phosphatase R181A/Q262A substrate-trapping double mutants with enhanced binding and decreased catalytic rates for identification of PTP substrates and inhibitors
INVENTOR(S): Zhang, Zhong-Yin; Xie, Laiping; Zhang, Yan-Ling
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 19 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2003170855	A1	20030911	US 2003-340288		20030110
PRIORITY APPLN. INFO.:			US 2002-347413P	P	20020114

AB The present invention provides use of protein tyrosine phosphatase R181A/Q262A substrate-trapping double mutants with enhanced binding and decreased catalytic rates for identification of PTP substrates and inhibitors. Protein engineering is performed upon protein tyrosine phosphatases (PTP) in which the invariant aspartate and glutamine residues are each replaced with alanine, wherein the replacement causes a redn. in catalytic rate (kcat) of the enzyme and an increase in substrate-binding affinity (Kd) of the enzyme. The present invention further provides methods for identifying a substrate of a PTP. Also provided are kits for identifying a substrate of a PTP. Addnl., the present invention provides methods for identifying an agent that alters interaction between a PTP and a substrate of the PTP. The invention also provides methods for reducing the activity of a substrate of a PTP.

L3 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:106025 HCAPLUS
DOCUMENT NUMBER: 128:177559
TITLE: Substrate-trapping protein tyrosine phosphatase mutants for identification of tyrosine-phosphorylated protein substrates and their clinical uses
INVENTOR(S): Tonks, Nicholas; Flint, Andrew J.
PATENT ASSIGNEE(S): Cold Spring Harbor Laboratory, USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9804712	A2	19980205	WO 1997-US13016	19970724
WO 9804712	A3	19980312		
W: CA, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5912138	A	19990615	US 1996-685992	19960725
CA 2262440	AA	19980205	CA 1997-2262440	19970724
AU 9859395	A1	19990216	AU 1998-59395	19970724
AU 728405	B2	20010111		
EP 918867	A2	19990602	EP 1997-937017	19970724

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2000515760	T2	20001128	JP 1998-508989	19970724
US 5951979	A	19990914	US 1998-144925	19980901
PRIORITY APPLN. INFO.:			US 1996-685992 A	19960725
			WO 1997-US13016 W	19970724

AB Novel protein tyrosine phosphatase mutants that are catalytically attenuated are prepd. by replacing the invariant aspartate residue with an amino acid residue to reduce the Kcat to <1 min-1. The mutation does not cause significant alteration of Km. Also described are methods of (1) identifying tyrosine phosphorylated proteins which complex with the described protein tyrosine phosphatase mutants; (2) identifying agents that interfere the interaction between a PTP and a tyrosine phosphatase; (3) reducing the transforming effects of oncogenes or the formation of signaling complexes assocd. with p130cas; and (4) reducing cytotoxic effects assocd. with PTP. Prepn. and characterization of PTP1B[D181A], PTP-PEST[D199A], and PTP-PEST[C231S] are also described.

L3 ANSWER 12 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:196438 BIOSIS
DOCUMENT NUMBER: PREV200100196438
TITLE: The receptor protein tyrosine phosphatase DEP-1 in endothelial cell signal transduction.
AUTHOR(S): Gruber, F. [Reprint author]; Koshelnick, Y. [Reprint author]; Prager, G. [Reprint author]; Hofer-Warbinek, R. [Reprint author]; Beckmann, R. [Reprint author]; Binder, B. R. [Reprint author]
CORPORATE SOURCE: Department of Vascular Biology and Thrombosis Research, University of Vienna, Vienna, Austria
SOURCE: Annals of Hematology, (2001) Vol. 80, No. Supplement 1, pp. A23. print.
Meeting Info.: 45th Annual Meeting of the Society for Thrombosis/Hemostasis Research. Duesseldorf, Germany. February 14-17, 2001. Society for Thrombosis/Hemostasis Research.
ISSN: 0939-5555.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Apr 2001
Last Updated on STN: 18 Feb 2002

L3 ANSWER 13 OF 19 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:523910 BIOSIS
DOCUMENT NUMBER: PREV199900523910
TITLE: Expression and regulation of protein tyrosine phosphatase (PTP) LAR in vascular smooth muscle cells (VSMC): Evidence for a role in proliferation.
AUTHOR(S): Bassett, Heather M.; Patterson, Cam; Runge, Marschall R.
CORPORATE SOURCE: Univ. Tex. Med. Branch, Galveston, TX, USA
SOURCE: Circulation, (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. I327. print.
Meeting Info.: 71st Scientific Sessions of the American Heart Association. Dallas, Texas, USA. November 8-11, 1998. The American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Dec 1999
Last Updated on STN: 5 Jun 2000

L3 ANSWER 14 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:743476 SCISEARCH
THE GENUINE ARTICLE: 845AV
TITLE: Regulated expression of the receptor-like tyrosine phosphatase CD148 on hemopoietic cells
AUTHOR: Lin J; Zhu J W; Baker J E; Weiss A (Reprint)
CORPORATE SOURCE: Univ Calif San Francisco, Dept Med, Rosalind Russell Med Res Ctr Arthritis, 533 Parnassus Ave, Room U-330, Box 0795, San Francisco, CA 94143 USA (Reprint); Univ Calif San Francisco, Dept Med, Rosalind Russell Med Res Ctr Arthritis, San Francisco, CA 94143 USA; Univ Calif San Francisco, Rosalind Russell Med Res Ctr Arthritis, Dept Microbiol & Immunol, San Francisco, CA 94143 USA; Univ Calif San Francisco, Rosalind Russell Med Res Ctr Arthritis, Biomed Sci Grad Program, San Francisco, CA 94143 USA; Univ Calif San Francisco, Rosalind Russell Med Res Ctr Arthritis, Howard Hughes Med Inst, San Francisco, CA 94143 USA
aweiss@medicine.ucsf.edu
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF IMMUNOLOGY, (15 AUG 2004) Vol. 173, No. 4, pp. 2324-2330.
ISSN: 0022-1767.
PUBLISHER: AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 32
ENTRY DATE: Entered STN: 10 Sep 2004
Last Updated on STN: 10 Sep 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB CD148 is a receptor-like protein tyrosine phosphatase expressed on a wide variety of cell types. Through the use flow cytometry and immunofluorescence microscopy on tissue sections, we examined the expression of CD148 on multiple murine hemopoietic cell lineages. We found that CD148 is moderately expressed during all stages of B cell development in the bone marrow, as well as peripheral mature B cells. In contrast, CD148 expression on thymocytes and mature T cells is substantially lower. However, stimulation of peripheral T cells through the TCR leads to an increase of CD148 expression. This up-regulation on T cells can be partially inhibited by reagents that block the activity of src family kinases, calcineurin, MEK, or PI3K. Interestingly, CD148 levels are elevated on freshly isolated T cells from MRL lpr/lpr and CTLA-4-deficient mice, two murine models of autoimmunity. Together, these expression data along with previous biochemical data suggest that CD148 may play an important regulatory role to control an immune response.

L3 ANSWER 15 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:245907 SCISEARCH
THE GENUINE ARTICLE: 779PP
TITLE: Site-selective regulation of platelet-derived growth factor beta receptor tyrosine phosphorylation by T-cell protein tyrosine phosphatase
AUTHOR: Persson C; Savenhed C; Bourdeau A; Tremblay M L; Markova B; Bohmer F D; Haj F G; Neel B G; Elson A; Heldin C H; Ronnstrand L; Ostman A; Hellberg C (Reprint)
CORPORATE SOURCE: Ludwig Inst Canc Res, Uppsala Branch, Ctr Biomed, Husargatan 3, Box 595, S-75124 Uppsala, Sweden (Reprint); Ludwig Inst Canc Res, Uppsala Branch, Ctr Biomed, S-75124 Uppsala, Sweden; McGill Univ, Ctr Canc, Montreal, PQ H3G 1Y6, Canada; Univ Jena, Fac Med, Inst Mol Cell Biol, D-07747 Jena, Germany; Harvard Univ, Sch Med, Beth Israel Deaconess Med Ctr, Dept Med, Div Hematol Oncol, Canc Biol

Program, Boston, MA 02215 USA; Weizmann Inst Sci, Dept Mol Genet, IL-76100 Rehovot, Israel
COUNTRY OF AUTHOR: Sweden; Canada; Germany; USA; Israel
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (MAR 2004) Vol. 24, No. 5, pp. 2190-2201.
ISSN: 0270-7306.
PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 41
ENTRY DATE: Entered STN: 19 Mar 2004
Last Updated on STN: 19 Mar 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The,platelet-derived growth factor (PDGF) beta receptor mediates mitogenic and chemotactic signals. Like other tyrosine kinase receptors, the PDGF beta receptor is negatively regulated by protein tyrosine phosphatases (PTPs). To explore whether T-cell PTP (TC-PTP) negatively regulates the PDGF beta receptor, we compared PDGF beta receptor tyrosine phosphorylation in wild-type and TC-PTP knockout (ko) mouse embryos. PDGF beta receptors were hyperphosphorylated in TC-PTP ko embryos. Fivefold-higher ligand-induced receptor phosphorylation was observed in TC-PTP ko mouse embryo fibroblasts (MEFs) as well. Reexpression of TC-PTP partly abolished this difference. As determined with site-specific phosphotyrosine antibodies, the extent of hyperphosphorylation varied among different autophosphorylation sites. The phospholipase Cgamma1 binding site Y1021, previously implicated in chemotaxis, displayed the largest increase in phosphorylation. The increase in Y1021 phosphorylation was accompanied by increased phospholipase Cgamma1 activity and migratory hyperresponsiveness to PDGF. PDGF beta receptor tyrosine phosphorylation in PTP-1B ko MEFs but not in PTPepsilon ko MEFs was also higher than that in control cells. This increase occurred with a site distribution different from that seen after TC-PTP depletion. PDGF-induced migration was not increased in PTP-1B ko cells. In summary, our findings identify TC-PTP as a previously unrecognized negative regulator of PDGF beta receptor signaling and support the general notion that PTPs display site selectivity in their action on tyrosine kinase receptors.

L3 ANSWER 16 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:854827 SCISEARCH
THE GENUINE ARTICLE: 604KU
TITLE: Evaluating function of transmembrane protein tyrosine phosphatase CD148 in lymphocyte biology
AUTHOR: Harrod T P; Justement L B (Reprint)
CORPORATE SOURCE: Univ Alabama, Dept Microbiol, Div Dev & Clin Immunol, Birmingham, AL 35294 USA (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: IMMUNOLOGIC RESEARCH, (2002) Vol. 26, No. 1-3, pp. 153-166
ISSN: 0257-277X.
PUBLISHER: HUMANA PRESS INC, 999 RIVERVIEW DRIVE SUITE 208, TOTOWA, NJ 07512 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 29
ENTRY DATE: Entered STN: 8 Nov 2002
Last Updated on STN: 8 Nov 2002
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The transmembrane protein tyrosine phosphatase CD148 is expressed on numerous cell types, including most cells of the hematopoietic lineage. CD148 has been shown to regulate density-dependent inhibition of cell growth as well as cellular differentiation in nonhematopoietic cells and has been shown to regulate signal transduction

processes in several nonlymphoid hematopoietic cell types. Analysis of CD 148 expression on lymphoid cells has demonstrated that CD148 is expressed at low levels on T cells and that it is upregulated in response to activation. Several groups have observed that CD148 negatively regulates T cell activation in response to crosslinking of the T cell antigen receptor, suggesting that it may play a role in feedback inhibition of the T cell immune response. In the B cell compartment, CD 148 expression appears to be restricted to the memory subpopulation, raising the possibility that it serves a unique function in these cells, which has yet to be determined. Recent studies have shown that CD148 interacts with the PDZ domain-containing protein syntenin, raising the possibility that its function or its localization with substrates in T and B cells may be controlled through this or a related interaction with another PDZ domain protein.

L3 ANSWER 17 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:421386 SCISEARCH
THE GENUINE ARTICLE: 424WZ
TITLE: The receptor protein tyrosine phosphatase DEP-1 in endothelial cell signal transduction
AUTHOR: Gruber F (Reprint); Koshelnick Y; Hofer-Warbinek R; Beckmann R; Kadl A; Prager G; Binder B R
CORPORATE SOURCE: Univ Vienna, Dept Vasc Biol & Thrombosis Res, A-1090 Vienna, Austria
COUNTRY OF AUTHOR: Austria
SOURCE: ARTERIOSCLEROSIS THROMBOSIS AND VASCULAR BIOLOGY, (APR 2001) Vol. 21, No. 4, pp. 697-697. MA 239.
ISSN: 1079-5642.
PUBLISHER: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
ENTRY DATE: Entered STN: 8 Jun 2001
Last Updated on STN: 8 Jun 2001

L3 ANSWER 18 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:824630 SCISEARCH
THE GENUINE ARTICLE: 249HH
TITLE: CD148, a new membrane tyrosine phosphatase involved in leukocyte function
AUTHOR: Gaya A (Reprint); Pirotto F; Palou E; Autschbach F; Del Pozo V; Sole J; Serra-Pages C
CORPORATE SOURCE: Fundacio Banc Sang & Teixits Illes Balears, Avinguda Gaspar Bennazar 73, Palma de Mallorca 07012, Spain (Reprint); Hosp Clin Barcelona, Serv Immunol, Barcelona, Spain
COUNTRY OF AUTHOR: Spain
SOURCE: LEUKEMIA & LYMPHOMA, (OCT 1999) Vol. 35, No. 3-4, pp. 237-+.
ISSN: 1042-8194.
PUBLISHER: HARWOOD ACAD PUBL GMBH, TAYLOR & FRANCIS GROUP, 325 CHESTNUT ST, 8TH FL, PHILADELPHIA, PA 19106 USA.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 27
ENTRY DATE: Entered STN: 1999
Last Updated on STN: 1999

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Protein tyrosine phosphatases play an essential role in the control of leucocyte cell growth an differentiation. Recently a new receptor type membrane tyrosine phosphatase named CD148 has been identified. This

molecule is present on the membrane of all the hematopoietic lineages as well as on several other cell types, mainly epithelial cells and its expression increases after cell activation. This molecule is able to act as a transducing molecule. Moreover, CD148 is able to modulate the signal transduction through the TCR/CD3 complex, in a manner similar to CD45, It has also been suggested that CD148 could be involved in mechanisms of differentiation and inhibition of cell growth. In addition, CD148 seems to be associated with a serine/threonine kinase in certain epitelial cell lines and leucocytes. Here, we review recent data on the expression and function of CD148 in both human, mouse and rat.

L3 ANSWER 19 OF 19 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:778076 SCISEARCH
THE GENUINE ARTICLE: 127GV
TITLE: Cutting edge: Negative regulation of human T cell activation by the receptor-type protein tyrosine phosphatase CD148
AUTHOR: Tangye S G (Reprint); Wu J; Aversa G; de Vries J E; Lanier L L; Phillips J H
CORPORATE SOURCE: DNAX Res Inst Mol & Cellular Biol Inc, Dept Immunobiol, 901 Calif Ave, Palo Alto, CA 94304 USA (Reprint); DNAX Res Inst Mol & Cellular Biol Inc, Dept Immunobiol, Palo Alto, CA 94304 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF IMMUNOLOGY, (15 OCT 1998) Vol. 161, No. 8, pp. 3803-3807.
ISSN: 0022-1767.
PUBLISHER: AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 31
ENTRY DATE: Entered STN: 1998
Last Updated on STN: 1998

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB T cell activation represents a balance between positive and negative signals delivered via distinct cell surface molecules. Many cytoplasmic protein tyrosine phosphatases are involved in regulating cellular responses by antagonizing the action of protein tyrosine kinases, CD148 is a receptor-type protein tyrosine phosphatase expressed by all human mononuclear cells. We have investigated the effect of CD148 on TCR-mediated activation of human T cells. Overexpression of wild-type, but not a phosphatase deficient, CD148 in Jurkat T cells inhibited TCR-mediated activation, evidenced by reduced expression of the early activation Ag CD69, inhibition of tyrosine phosphorylation of many intracellular proteins including the critical protein tyrosine kinase ZAP-70, and impairment of mitogen-activated protein kinase activation. Taken together, these results suggest that CD148 is an important phosphatase involved in negatively regulating the proximal signaling events during activation of Ag-specific T cells.

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L1 318 S (DENSITY ENHANCED POLYPEPTIDE-1 OR DEP-1)
L2 130 DUP REM L1 (188 DUPLICATES REMOVED)
L3 19 S L2 AND SIGNAL TRANSDUCTION
L4 0 S L3 AND DEP-1 SUBSTRATE POLYPEPTIDE

=> s l3 and 1990-2002

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L5            0 L3 AND 1990-2002

=> s l3 and 1990-2002/py
L6           11 L3 AND 1990-2002/PY

=> d his

     (FILE 'HOME' ENTERED AT 13:43:38 ON 03 NOV 2006)

     FILE 'MEDLINE, HCAPLUS, BIOSIS, BIOTECHDS, SCISEARCH, EMBASE' ENTERED AT
     13:45:00 ON 03 NOV 2006
L1           318 S (DENSITY ENHANCED POLYPEPTIDE-1 OR DEP-1)
L2           130 DUP REM L1 (188 DUPLICATES REMOVED)
L3            19 S L2 AND SIGNAL TRANSDUCTION
L4             0 S L3 AND DEP-1 SUBSTRATE POLYPEPTIDE
L5             0 S L3 AND 1990-2002
L6            11 S L3 AND 1990-2002/PY

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☐ 1. Document ID: US 20060198847 A1

L3: Entry 1 of 10 File: PGPB Sep 7, 2006

PGPUB-DOCUMENT-NUMBER: 20060198847
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060198847 A1

TITLE: Modulation of endothelial cell surface receptor activity in the regulation of angiogenesis

PUBLICATION-DATE: September 7, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Daniel; Thomas O.	Nashville	TN	US
Takahashi; Takamune	Nashville	TN	US
Mernaugh; Raymond	Nashville	TN	US

US-CL-CURRENT: 424/155.1; 530/388.8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20060014180 A1

L3: Entry 2 of 10 File: PGPB Jan 19, 2006

PGPUB-DOCUMENT-NUMBER: 20060014180
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060014180 A1

TITLE: Human phosphatase RET31, and variants thereof

PUBLICATION-DATE: January 19, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Jackson; Donald G.	Lawrenceville	NJ	US
Ramanathan; Chandra S.	Wallingford	CT	US
Feder; John N.	Belle Mead	NJ	US
Mintier; Gabe	Hightstown	NJ	US
Lee; Liana	North Brunswick	NJ	US
Nelson; Thomas C.	Lawrenceville	NJ	US
Siemers; Nathan	Pennington	NJ	US
Bol; David	Langhorne	PA	US

Suchard; Suzanne	Wilmington	DE	US
Schieven; Gary	Lawrenceville	NJ	US
Finger; Joshua	San Marcos	CA	US
Todderrud; C. Gordon	Newtown	PA	US
Bassolino; Donna	Hamilton	NJ	US
Krystek; Stanley	Ringoes	NJ	US
Banas; Dana	Hamilton	NJ	US
McAtee; Patrick	Pennington	NJ	US

US-CL-CURRENT: 435/6; 435/320.1, 435/325, 435/69.1, 530/350, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 3. Document ID: US 20040161821 A1

L3: Entry 3 of 10 File: PGPB Aug 19, 2004

PGPUB-DOCUMENT-NUMBER: 20040161821
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040161821 A1

TITLE: DEP-1 receptor protein tyrosine phosphatase interacting proteins and related methods

PUBLICATION-DATE: August 19, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Palka-Hamblin, Helena L.	Rego Park	NY	US
Tonks, Nicholas K.	Huntington	NY	US

US-CL-CURRENT: 435/69.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 20040038207 A1

L3: Entry 4 of 10 File: PGPB Feb 26, 2004

PGPUB-DOCUMENT-NUMBER: 20040038207
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040038207 A1

TITLE: Gene expression in bladder tumors

PUBLICATION-DATE: February 26, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Orntoft, Torben F.	Aabyhoj		DK

US-CL-CURRENT: 435/6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 5. Document ID: US 6936417 B2

L3: Entry 5 of 10 File: USPT Aug 30, 2005

US-PAT-NO: 6936417
DOCUMENT-IDENTIFIER: US 6936417 B2

TITLE: Gene expression in bladder tumors

DATE-ISSUED: August 30, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Orntoft; Torben F.	Aabyhoj			DK

US-CL-CURRENT: 435/6; 435/91.2, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 6. Document ID: US 6335170 B1

L3: Entry 6 of 10 File: USPT Jan 1, 2002

US-PAT-NO: 6335170
DOCUMENT-IDENTIFIER: US 6335170 B1

TITLE: Gene expression in bladder tumors

DATE-ISSUED: January 1, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Orntoft; Torben F.	DK 8230 Aabyhoj			DK

US-CL-CURRENT: 435/6; 435/91.1, 435/91.2, 536/23.1, 536/24.3, 536/24.31, 536/24.33

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 7. Document ID: US 6248327 B1

L3: Entry 7 of 10 File: USPT Jun 19, 2001

US-PAT-NO: 6248327
DOCUMENT-IDENTIFIER: US 6248327 B1

TITLE: Modulation of endothelial cell surface receptor activity in the regulation of angiogenesis

DATE-ISSUED: June 19, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Daniel; Thomas O.	Nashville	TN		
Takahashi; Takamune	Nashville	TN		

US-CL-CURRENT: 424/143.1; 424/152.1, 424/156.1, 514/12, 514/2, 514/21, 530/388.22, 530/388.85

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 8. Document ID: WO 2004048549 A2

L3: Entry 8 of 10 File: EPAB Jun 10, 2004

PUB-NO: WO2004048549A2
DOCUMENT-IDENTIFIER: WO 2004048549 A2
TITLE: DEP-1 RECEPTOR PROTEIN TYROSINE PHOSPHATASE INTERACTING PROTEINS AND RELATED METHODS

PUBN-DATE: June 10, 2004

INVENTOR-INFORMATION:

NAME	COUNTRY
PALKA-HAMBLIN, HELENA L	US
TONKS, NICHOLAS K	US

INT-CL (IPC): C12N 0/
EUR-CL (EPC): C07K014/705

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 9. Document ID: EP 1576139 A2, WO 2004048549 A2, US 20040161821 A1, AU 2003298761 A1

L3: Entry 9 of 10 File: DWPI Sep 21, 2005

DERWENT-ACC-NO: 2004-450367
DERWENT-WEEK: 200562

TITLE: New isolated complex comprises Density Enhanced Phosphatase-1 (DEP-1) polypeptide, useful for manipulating biological signal transduction pathways, or determining additional molecular components of the pathways

INVENTOR: PALKA-HAMBLIN, H L; TONKS, N K

PRIORITY-DATA: 2002US-429746P (November 26, 2002), 2003US-0723606 (November 26, 2003)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1576139 A2	September 21, 2005	E	000	C12N001/00
WO 2004048549 A2	June 10, 2004	E	130	C12N000/00
US 20040161821 A1	August 19, 2004		000	C12P021/06
AU 2003298761 A1	June 18, 2004		000	C12N000/00

INT-CL (IPC): C12N 0/00; C12N 1/00; C12P 21/06

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 10. Document ID: WO 200164750 A2, AU 200139898 A, EP 1261644 A2, JP 2004507210 W, MX 2002008462 A1, US 20060198847 A1

L3: Entry 10 of 10 File: DWPI Sep 7, 2001

DERWENT-ACC-NO: 2001-570681
DERWENT-WEEK: 200659

TITLE: Novel antibody for modulating angiogenesis and endothelial cell migration and proliferation, binds endothelial cell receptor tyrosine phosphatase/density enhanced phosphatase-1

INVENTOR: DANIEL, T O; MERNAUGH, R ; TAKAHASHI, T

PRIORITY-DATA: 2000US-0516728 (March 1, 2000), 1998US-0152160 (September 11, 1998), 2006US-0416492 (May 2, 2006)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200164750 A2	September 7, 2001	E	110	C07K016/00
AU 200139898 A	September 12, 2001		000	C07K016/00
EP 1261644 A2	December 4, 2002	E	000	C07K016/28
JP 2004507210 W	March 11, 2004		184	C12N015/09
MX 2002008462 A1	May 1, 2004		000	C07K016/00
US 20060198847 A1	September 7, 2006		000	A61K039/395

INT-CL (IPC): A01K 67/027; A61K 35/74; A61K 35/76; A61K 38/00; A61K 38/17; A61K 39/395; A61K 48/00; A61P 9/10; A61P 17/06; A61P 19/10; A61P 27/02; A61P 29/00; A61P 35/00; A61P 35/04; C07K 1/22; C07K 16/00; C07K 16/18; C07K 16/28; C07K 16/30; C07K 16/46; C12N 1/15; C12N 1/19; C12N 1/21; C12N 5/08; C12N 5/10; C12N 15/09; C12P 21/08; C12Q 1/02; C12Q 1/42; C12Q 1/68; G01N 30/48; G01N 30/88; G01N 33/15; G01N 33/50; G01N 33/53

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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Terms	Documents
Density Enhanced Phosphatase-1	10

Display Format: - Change Format

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L3: Entry 7 of 10 File: USPT Jun 19, 2001

US-PAT-NO: 6248327
DOCUMENT-IDENTIFIER: US 6248327 B1

TITLE: Modulation of endothelial cell surface receptor activity in the regulation of angiogenesis

DATE-ISSUED: June 19, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Daniel; Thomas O.	Nashville	TN		
Takahashi; Takamune	Nashville	TN		

US-CL-CURRENT: 424/143.1; 424/152.1, 424/156.1, 514/12, 514/2, 514/21, 530/388.22, 530/388.85

CLAIMS:

What is claimed is:

1. A method of modulating angiogenesis in a vertebrate subject, the method comprising administering to the vertebrate subject an ECRTP/DEP-1 receptor activity-modulating amount of a composition, whereby an ECRTP/DEP-1 receptor within the vertebrate subject is contacted by the composition; and modulating angiogenesis through the contacting of the ECRTP/DEP-1 receptor with the composition.

2. The method of claim 1, wherein the composition comprises a monoclonal antibody which selectively binds the ECRTP/DEP-1 receptor.

3. The method of claim 2, wherein the monoclonal antibody is monoclonal antibody ECRTPAb-1, having a molecular weight of about 150 kDa and which selectively binds to an ectodomain of the ECRTP/DEP-1 receptor.

4. The method of claim 3, wherein the ECRTP/DEP-1 receptor activity-modulating amount of the monoclonal antibody ranges from about 0.1 to about 300 milligrams per kilogram body weight of the vertebrate subject.

5. The method of claim 4, wherein the ECRTP/DEP-1 receptor activity-modulating amount of the monoclonal antibody ranges from about 0.2 to about 200 milligrams per kilogram body weight of the vertebrate subject.

6. The method of claim 5, wherein the ECRTP/DEP-1 receptor activity-modulating amount of the monoclonal antibody ranges from about 0.5 to about 20 milligrams per kilogram body weight of the vertebrate subject.

7. The method of claim 3, wherein the monoclonal antibody is further characterized as having the immunoreaction characteristics of a monoclonal antibody produced by a hybridoma cell line having ATCC accession number HB12570.

8. The method of claim 7, where the monoclonal antibody is monoclonal antibody produced by

a hybridoma cell line having ATCC accession number HB12570.

9. The method of claim 2, wherein the antibody is humanized.

10. The method of claim 9, wherein the humanized antibody is humanized monoclonal antibody ECRTPAb-1, having a molecular weight of about 150 kDa and which preferentially binds to an ectodomain of the ECRTP/DEP-1 receptor.

11. The method of claim 10, wherein the humanized antibody is further characterized as having the immunoreaction characteristics of a monoclonal antibody produced by a hybridoma cell line having ATCC accession number HB12570.

12. The method of claim 11, where the monoclonal antibody is monoclonal antibody produced by a hybridoma cell line having ATCC accession number HB12570.

13. The method of claim 1, wherein the administering is selected for the group consisting of intravenous administration, intrasynovial administration, transdermal administration, intramuscular administration, subcutaneous administration and oral administration.

14. The methods of claim 1, wherein the administering is conducted in conjunction with chemotherapy.

15. The method of claim 1, wherein the vertebrate subject is a mammal.

16. The method of claim 15, wherein the mammal is a human.

17. The method of claim 1, wherein said angiogenesis comprises angiogenesis in a solid tumor in a patient, and wherein an ECRTP/DEP-1 receptor expressed on the surface of vascular endothelial cells involved in the angiogenesis is contacted by the composition resulting in inhibition in the blood supply to tissue of the solid tumor.

18. The methods of claim 17, wherein the administering is conducted in conjunction with chemotherapy.

19. The method of claim 17, wherein the patient is a human.

20. The method of claim 1, wherein the angiogenesis comprises angiogenesis in an inflamed tissue of a patient and wherein an ECRTP/DEP-1 receptor expressed on the surface of vascular endothelial cells involved in the angiogenesis in the inflamed tissue is contacted by the modulator resulting in inhibition in the blood supply to the inflamed tissue.

21. The method of claim 20, wherein the patient is a human.

22. A method of modulating angiogenesis in a vertebrate subject, the method comprising administering to the vertebrate subject an ECRTP/DEP-1 receptor activity inhibiting amount of an antibody, wherein the antibody selectively binds the ECRTP/DEP-1 receptor, whereby an ECRTP/DEP-1 receptor within the vertebrate subject is contacted by the antibody; and inhibiting angiogenesis through the contacting of the ECRTP/DEP-1 receptor with the antibody.

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☐	L2	DEP-1 substrate polypeptide	3
☐	L1	DEP substrate polypeptide	0

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	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L4	L1 and biological signal	3
☐	L3	L1 and transduction?	0
☐	L2	L1 and tranduction?	0
☐	L1	density enhanced phosphatase-1	10

END OF SEARCH HISTORY